THE EFFECT OF TREADMILL WALKING CONDITIONS (LEVEL, UPHILL, DOWNHILL) ON QUADRICEPS EMG AMPLITUDES IN INDIVIDUALS WITH ANTERIOR CRUCIATE LIGAMENT RECONSTRUCTION AND HEALTHY CONTROLS

Jamie Albrecht

A thesis submitted to the faculty of the University of North Carolina at Chapel Hill in partial fulfillment of the requirements for the degree of Master of Arts in the Department of Exercise and Sport Science (Athletic Training).

Chapel Hill
2020

Approved by:

Troy Blackburn

Brian Pietrosimone

Derek Dewig

ABSTRACT

Jamie Albrecht: The effect of treadmill walking conditions (level, uphill, downhill) on quadriceps EMG amplitudes in individuals with anterior cruciate ligament reconstruction and healthy controls
(Under the direction of Troy Blackburn)

Quadriceps dysfunction leads to aberrant gait biomechanics that are hypothesized to contribute to post-traumatic knee osteoarthritis (PTOA) following anterior cruciate ligament reconstruction (ACLR). Graded walking (uphill and downhill) may exacerbate abnormalities that otherwise go unnoticed. The purpose of this study was to compare quadriceps electromyography (EMG) amplitudes between level, uphill, and downhill walking conditions in individuals at least one year post-ACLR. Quadriceps EMG was sampled in 24 ACLR and 27 healthy controls during treadmill walking. Quadriceps EMG centered on heel strike differed up to 25% between groups, though not significant. Significant correlations were observed between involved limb: peak torque and uphill EMG change scores, and rate of torque development 100ms after activation and uphill EMG change scores centered on heel strike and at 50% of stance. Quadriceps dysfunction remains present in ACLR individuals compared to healthy controls when walking uphill. Current rehabilitation may be insufficient to address this specific condition.

TABLE OF CONTENTS

LIST OF FIGURES vi

LIST OF TABLES vii

LIST OF ABBREVIATIONS viii

CHAPTER 1 INTRODUCTION 1

Specific Aims & Hypotheses 5

CHAPTER 2 LITERATURE REVIEW 7

Prevalence and consequences 7

Gait biomechanics following ACLR 8

Quadriceps function during gait 11

Biomechanics in graded gait conditions 12

Biomechanics in graded gait conditions following ACLR 14

Measures of self-report function after ACLR 15

Conclusions 16

CHAPTER 3 METHODS 18

Design 18

Procedures 18

Gait Biomechanics Assessment 19

Quadriceps Function Assessment 20

Statistical Analysis 21

CHAPTER 4 RESULTS 23

CHAPTER 5 DISCUSSION 27

REFERENCES 33

LIST OF FIGURES

Figure 1 – Average Quadriceps EMGP 24

Figure 2 – Average Quadriceps EMG50% 24

Figure 3 – Average Quadriceps EMGC... 25

LIST OF TABLES

Table 1 - Average demographic information of subjects.. 23

Table 2 – Estimated marginal means 25

Table 3 - Statistical results of correlations.. 26

LIST OF ABBREVIATIONS

ACL	Anterior cruciate ligament
ACLR	Anterior cruciate ligament reconstruction
DC	Direct current
EMG	Electromyography
IKDC	International Knee Documentation Committee
MOI	Mechanism of injury
MVIC	Maximal voluntary isometric contractions
OA	Osteoarthritis
PT	Peak torque
PTOA	Post-traumatic osteoarthritis
RTD	Rate of torque development
TKA	Total knee arthroplasty
vGRF	Vertical ground reaction forces
WBV	Whole body vibration
WOMAC	Western Ontario McMaster Universities Osteoarthritis Index

CHAPTER 1

INTRODUCTION

Anterior cruciate ligament (ACL) injury is one of the most common injuries in sports involving fast paced change of direction.[1,2] An estimated 200,000 ACL injuries occur annually in the U.S.[1] Females in particular are at a heightened risk with the combined college basketball and soccer female ACL rupture rate alone as high as 30% per 1,000 exposures.[3] ACL injuries create both physical and financial challenges for patients. ACL reconstruction (ACLR) costs are between $5,000 to $17,000 per patient, but long-term costs may reach as much as $38,000 per patient,[1,4] and non-surgical ACL rehabilitation costs are even greater.[4] Post-traumatic knee osteoarthritis (PTOA) risk is linked to ACL injury and contributes to lifelong health effects. Within 10 to 20 years, individuals with ACL injury are 60%-90% more likely to develop PTOA compared to individuals without ACL injuries.[5] This heightened risk for PTOA leads to additional financial burdens and a decreased quality of life, and may lead to other pathologies at the hip, knee, or ankle.[2,6]

Aberrant gait biomechanics are hypothesized to be a primary contributor to the development of PTOA. Radiographic signs of PTOA are present 5 years following ACLR in individuals who walk with smaller knee flexion moments and angles.[6,7] Therefore, these changes in sagittal plane loading and motion may be linked to the development of PTOA and long-term health consequences. Improper mechanics may affect the position of the knee upon loading causing changes in contact pressures on knee cartilage[8] and a decreased ability of the quadriceps muscles to dissipate force.

Quadriceps dysfunction is common following ACLR and is characterized by muscle inhibition, activation failure, and weakness.[9,10] Dysfunction may arise from muscle atrophy, fatty infiltration of the muscle, arthrogenic inhibition, or another form of neuromuscular deficit. This can lead to decreased force dissipation at the knee, increased joint surface damage, and increased impulsive loading which has been connected to PTOA development.[11–14] The quadriceps act as a shock absorber to dissipate ground impact forces during gait,[11] thus weakness may contribute to aberrant gait mechanics due to the inability to adequately produce a sufficient sagittal plane moment at the knee. Lewek, *et al.*[15] found that during overground walking, ACLR individuals with quadriceps weakness displayed smaller sagittal plane angles and moments during weight acceptance than uninjured subjects, but ACLR individuals with acceptable quadriceps strength walked similarly to uninjured controls. Studies utilizing quadriceps electromyography (EMG) have reported increased quadriceps neural activation in ACLR individuals compared to healthy controls during the early stance/loading phase of gait followed by a decrease in the full stance phase of gait.[16] However, these studies report that these EMG values in individuals with ACLR generally tend to approach normal values when compared to healthy controls.[16,17] Increased EMG activity upon loading/early stance may indicate compensation for lack of quadriceps strength and ability to attenuate the force created during heel strike.

Alterations in gait biomechanics, such as changes in sagittal and frontal plane moments and joint loading rate[2] have been found within the first year post-ACLR likely due in large part to effusion, pain, and muscle weakness. However current research is inconclusive in regards to biomechanics in individuals who are greater than one year post-ACLR.[2,7,11] Noehren, *et al.*[18] found significantly lower knee extensor moments, but no statistical differences in knee flexion angle between individuals with ACLR and healthy controls. On the contrary, studies included in the systematic review by Hart, *et al.*[12] found that after one year post-ACLR there is a lower knee flexion angle,[19,20] but no differences in knee flexion[21,22] and adduction moments[21,23–26] during overground walking. Other studies reported a combination of the above findings stating that individuals who went on to develop PTOA demonstrated decreased peak knee flexion moments, decreased peak knee flexion angles, and slightly increased peak adduction moments at the five-year time point.[6,7] The majority of current studies have evaluated gait biomechanics following ACLR during level walking, but this may not be able to elucidate long-term gait alterations due to the simplicity of the task and ease of completion after the resolution of acute signs and symptoms. The assessment of gait in graded conditions may be better served to evaluate differences in long-term joint loading, as uphill and downhill walking necessitate a greater internal knee extension moment than level walking.[27,28] Since sagittal plane movement is greatly influenced by quadriceps function, investigation of more challenging conditions may reveal deficiencies in these muscles. In stair climbing, one study found that the peak knee external flexion moment of the ACLR limb was significantly lower than the contralateral limb during ascent and descent.[21] Compensatory increased loading in the contralateral knee of ACLR individuals was also noted, likely due to lower loading of the ACLR knee.[21] Activities of daily living require ascending and descending on graded surfaces and may exacerbate gait differences that may exist in ACLR individuals greater than one year post-surgery that may otherwise go undetected.

Quadriceps EMG may be a valuable tool to help identify quadriceps function deficits. EMG measures the amount of neural activation in a target tissue by measuring action potential generation. Increased EMG signals, particularly in an injured limb, may indicate the need to activate more of the motor neuron pool to complete a given task than would be needed in an uninjured limb. Therefore, differences in EMG during various tasks may suggest dysfunction within the tissue itself or within neurological pathways. Identification of quadriceps activation changes in ACLR individuals at later post-operative time points may allow clinicians to focus rehabilitation strategies to create better overall patient outcomes and mitigate PTOA risk. Quadriceps EMG research among healthy and ACLR patients across different graded conditions has also been scarcely researched, but some studies have analyzed EMG during treadmill,[29,30] ramp,[17,31] and stair[17] conditions. These studies have indicated increased quadriceps EMG activity during uphill activities compared to level walking due to increased demands of the task as well as overcoming the co-contraction of the hamstrings.[17,31]

It is important to evaluate the influence of surface grade on quadriceps activation as quadriceps dysfunction is common following ACLR and is related to aberrant overground gait biomechanics linked to PTOA. This would allow researchers to better understand how activities of daily living, particularly repetitive activities such as walking on various grades, are affected following ACLR. Quadriceps activation alterations may be an important aspect to understanding PTOA and dysfunction of the quadriceps muscles may have significant, life-long effects on ACLR patients. Comparing differences in gait biomechanics over different conditions between ACLR and healthy controls is important as it may help elucidate changes in gait biomechanics linked to PTOA development.[2] Furthermore, current literature is ambiguous regarding how gait changes after one year post-ACLR. Alterations in knee joint moments, angles, and loading rates are not uniformly reported in the literature, and therefore warrant further investigation. Understanding how gait biomechanics are altered in graded conditions may provide new information as to why these changes are present years after surgery and how they affect the development of PTOA. The purpose of this study was to determine how quadriceps EMG amplitudes are affected among different graded walking conditions in ACLR individuals that are at least one year post-surgery. It is imperative that quadriceps activation and gait changes in individuals with ACLR are fully understood as that may allow clinicians to design better rehabilitation strategies for patients in the short term and long term, thereby improving patient outcomes.

Specific Aims & Hypotheses

Specific Aim 1: To compare the effect of treadmill walking condition (level, uphill, downhill) on quadriceps EMG amplitudes between individuals with ACLR and healthy controls.

Hypothesis: We hypothesize that the uphill and downhill conditions will result in greater increases in EMG amplitudes relative to level walking in ACLR individuals compared to healthy controls.

Specific Aim 2: To evaluate the relationship between quadriceps function and changes in quadriceps EMG amplitudes between different gait conditions in individuals with ACLR.

Hypothesis: We hypothesize that individuals with poorer quadriceps function (e.g. lower peak torque and rate of torque development) will display larger increases in quadriceps EMG amplitude outcomes when walking uphill/downhill compared to level walking.

CHAPTER 2

LITERATURE REVIEW

Prevalence and consequences

Anterior cruciate ligament (ACL) injuries are among the most common injuries in sport, particularly among young individuals involved in fast paced, change of direction sports.[1,2] There are approximately 200,000 ACL injuries annually in the U.S.[4] Not only do these injuries cause long-term health effects, but the prescribed rehabilitation and treatment incurs great financial burden. Estimates of lifetime burden costs in the U.S. are approximately $7.6 billion annually for anterior cruciate ligament reconstruction (ACLR), and $17.7 billion annually for non-operative treatment.[4] ACLR costs range from $5,000-$17,000 per surgical procedure, but estimated long-term societal costs may reach $38,000 per patient.[1,4] Individuals who choose the non-surgical option incur lifetime costs of approximately $88,000.[4]

Additionally, ACL injury often leads to debilitating concomitant health issues leading to a decreased quality of life. Post-traumatic osteoarthritis (PTOA) is one of the most notable physical consequences following ACL injury. The risk of developing this injury rise drastically after ACL injury.[4,5] Individuals are 4 times more likely to develop PTOA following ACLR suggesting that previous knee injury is a significant risk factor for early PTOA development.[32,33] Approximately 60%-90% of individuals develop PTOA within the first 10 to 20 years following injury.[5] Mather *et al.*[4] described that of the 200,000 annual ACL injuries, 118,000 would develop radiographic osteoarthritis over their lifetime. Of those, 31,600 would develop symptoms and 25,800 would require total knee arthroplasty.[4] In contrast, if all of those individuals received structured rehabilitation without ACLR as treatment, 140,000 would develop radiographic osteoarthritis over their lifetime. Of those, 38,000 would develop symptoms and 30,800 would need a total knee arthroplasty (TKA).[4]

ACL injuries are a frequent occurrence in sport and can have lifelong health consequences. These injuries not only cause physical problems, but also pose potential financial burdens and a decreased quality of life. Additionally, individuals are extremely likely to develop secondary injuries, most notably PTOA. Investigating prophylactic and therapeutic methods to manage this problem post injury are key to decreasing the likelihood of these negative effects.

Gait biomechanics following ACLR

Aberrant gait biomechanics are common following ACLR.[2] These changes occur initially after ACLR and evolve throughout the recovery process.[12] Therefore, stress and location of stress placed along the lower extremity may be altered with every step ACLR individuals take. The repetitive nature of walking leads to the inference that the effects of altered may accumulate over time. Improper joint loading, altered joint moments, and suboptimal force dissipation in ACLR individuals may lead to further health issues across their lifetime.

Gait abnormalities can further contribute to an increased risk of developing PTOA.[2] ACLR limbs show a greater rate of loading compared to both healthy and contralateral limbs.[11,18,34] Abnormal loading may contribute to mechanical damage by placing excessive stress on the articular cartilage of the knee contributing to the early development of PTOA.[2,7,12] According to Pietrosimone *et al*.[35] differences exist when comparing lower-extremity loading and time post ACLR. Symptomatic individuals less than 12 months removed from ACLR showed less vertical ground reaction forces (vGRF) during the first and last thirds of stance, but greater vGRF during the midstance phase of gait in comparison to asymptomatic individuals.[35] Therefore, symptomatic individuals <12 months post-ACLR tended to underload the ACLR-limb during the weight acceptance and propulsive phases of stance. Conversely, symptomatic individuals >24 months post-ACLR showed greater vGRF during the first and last thirds of stance, but lesser vGRF during midstance in comparison to asymptomatic individuals. This demonstrated that symptomatic individuals >24 months post-ACLR tended to overload the ACLR-limb during the weight acceptance and propulsion phases of stance.[35] These findings suggest that mechanical loading of the lower extremity is associated with symptoms and time post-ACLR. Additionally, these findings would indicate that rehabilitation goals should be considered more carefully depending on the time elapsed since ACLR. Continued research to understand long term factors following ACL injury and their association with resultant PTOA are necessary to improve patient outcomes. Long-term knee health may be affected significantly due to prolonged alterations in joint loading.[35]

In a systematic review conducted by Hart *et al.*,[12] biomechanical changes, such as greater peak knee flexion angle, greater peak knee flexion moment, and lower knee extension moment were the most prominent in the sagittal plane up to six months after ACLR. Within one year post-surgery, peak adduction moments have been shown to be significantly less in ACLR limbs when compared to controls.[2] In contrast, Miyazaki *et al.*[36] found that larger knee adduction moments were present within one year of injury and were associated with greater medial knee loading, suggesting an increased risk of medial compartment PTOA. Individuals show a smaller peak flexion angle greater than one year post ACLR, but no differences in external flexion moment.[12] At the five year postoperative time point, individuals who went on to develop PTOA demonstrated decreased peak knee flexion moments, decreased peak knee flexion angles, and slightly increased peak adduction moments.[6,7] This may be attributed to quadriceps weakness.[6] Peak knee flexion angle was restored and similar to that of the contralateral limb up to six years post-ACLR.[2] However, this was not true for internal knee extension moment during walking which displayed lower moments.[2]

During an optimal landing task, the knee is allowed to go through significant flexion controlled by eccentric quadriceps activity with little hamstring activity.[37] However, greater co-contraction of the quadriceps and hamstrings in ACLR individuals has been observed compared to healthy controls.[38] Observing heightened co-contraction post ACLR may be a sign of altered quadriceps function causing a decreased ability of the quadriceps to produce eccentric forces. Those with greater co-contraction measures exhibited increased tibiofemoral compressive loads and smaller tibiofemoral contact forces.[39,40] Blackburn *et al.*[41] found that ACLR limbs displayed greater co-activation during the preparatory and heel strike phases of gait when compared to the contralateral side and to healthy controls as well.[41] Lesser knee flexion displacement, lesser internal knee extension moment, and greater internal knee valgus moment were also revealed.

These factors together describe the ways in which knee cartilage is loaded improperly and suggest how cartilage degeneration leading to PTOA is heightened.

Quadriceps function during gait

Quadriceps dysfunction is often a difficult, lingering issue following ACLR. Quadriceps dysfunction can involve decreased strength, neuromuscular capability, and coordination and can be a result of muscle atrophy and arthrogenic muscle inhibition following ACLR. The quadriceps act as a shock absorber to dissipate ground impact forces during gait[11] and in the presence of dysfunction, force dissipation during gait can become problematic.[10,11] During the early stance of walking and jogging, ACLR individuals with quadriceps strength <90% of the uninvolved side had reduced knee angles and moments.[15] In contrast, knee angles and moments of ACLR individuals with quadriceps strength >90% of the uninvolved side were indistinguishable from an uninjured group.[15] There was a significant effect between early stance phase knee angles and moments and quadriceps strength during both walking and jogging.[15] This dysfunction can cause impulsive loading which has been connected with PTOA development.[11,18] Animal studies have demonstrated cartilage damage and chondrocyte death at higher rates of loading.[13] Therefore, it is plausible that impulsive loading of the knee in humans has a similar effect. Impulsive loading may be one aspect of the multifaceted issue of PTOA and may be moderated by quadriceps function.

EMG measures the electrical activity of the target muscle and therefore provides information about the activation of that muscle. EMG amplitudes supply data regarding the extent to which activation is achieved. As motor units in the target tissue are recruited, the EMG electrodes measure the action potentials that are created. Greater recruitment of motor units corresponds to greater EMG signal. This allows researchers to compare EMG measures among injured and non-injured individuals as well as compare injured individuals to an uninvolved side when applicable. EMG amplitudes are important measures when comparing ACLR patients to their contralateral leg or to non-ACLR individuals to determine if there are changes in quadriceps function during numerous functional tasks including treadmill walking, incline or decline walking, stair climbing, or hopping. Deficits may indicate dysfunction within the tissue itself or neurologically. This information, combined with kinematic and kinetic data and self-reported outcomes, may provide the researcher with potential conclusions as to why ACLR patients have altered gait, joint loading, and a higher risk for the development of PTOA.

Biomechanics in graded gait conditions

Human locomotion depends on the ability of the neuromuscular system to recruit leg muscles in response to the changing environment.[42] Everyday, humans encounter uphill and downhill walking conditions. Analyzing biomechanics of gait in different walking positions and speed may give valuable insight into how the nervous system controls movement.[27] Compared to level walking, humans need net positive work to walk uphill and net negative work to walk downhill in order to raise or lower the body's center of mass.[43] When walking at different grades, individual leg mechanical work during a stride changes and joint work also changes during the stance phase.[44]

During level walking, the knee joint contributes approximately 20% of total positive joint work.[45] During uphill and downhill walking, the knee joint contributes approximately 17%-28% of total positive joint work.[45] The greatest difference in positive joint work for the knee occurred between level walking and 18° of incline walking. The knee produces 35%-70% of total negative work and is most predominant during downhill walking.[45] Franz *et al.*[43] showed that during uphill walking in the double support phase, the trailing leg of gait performed greater positive work and during downhill walking the leading leg performed greater negative work. Knee joint peak flexion moment and peak extension moment increased on level ground with faster walking speed, but decreased with increased slope.[44]

Muscles acting at the knee absorb 40% more peak negative power when walking downhill at 21.5° compared to level ground.[31] This is likely due to leg extensor muscles meeting the demands of incline or decline walking by producing greater concentric force for uphill activity and greater eccentric force for downhill activity.[31] During uphill walking, hip, knee, and ankle extensor muscle activations increased while walking at the same speed, but during downhill walking only the knee extensor muscle activation increased while walking at the same speed.[31] Alexander *et al.*[45] showed knee positive work increased up to six times during uphill walking compared to level walking, and negative and absolute knee work increased seven times compared to level walking.[45] However, Montgomery and Grabowski[44] did not find a significant difference in positive and negative work contribution in the knee during uphill and downhill slopes in healthy individuals. Peak knee joint flexion power increased on level ground with faster walking speed, but remained constant with change in slope. Knee joint extension power decreased on level ground with faster walking speed increased with increasing slope.[44] As the grade of incline increases during uphill walking, there is also an increase in quadriceps muscle activity.[29]

Lay, *et al.*[27] found that during downslope walking, internal knee extensor moments increased for the majority of the stance phase. The authors hypothesized that this finding suggests a different neural control strategy might be used during downhill walking.[27] Knee moment patterns in upslope walking were found to be similar to that of level walking.[27] Franz and Kram[42] found that knee extensor activity was increased significantly for both uphill and downhill walking. In steeper uphill walking there was a greater increase in vastus medialis activity compared to rectus femoris activity.[42] This was significantly different from walking downhill, in which the two muscles responded similarly.[42]

Biomechanics in graded gait conditions following ACLR

Many current studies have evaluated gait biomechanics one year or more following ACLR during level walking, but there is paucity in regards to graded gait conditions. After one year post-op, literature suggests that gait biomechanics in ACLR individuals seem to normalize over level walking. However, graded walking conditions may exacerbate and detect biomechanical differences more so than level walking alone at this time point. As activities of daily living require ascending and descending on a graded surface (uphill and downhill), it is important to understand the effect these conditions have on underlying dysfunction. Much of the current literature surrounding individuals with PTOA and ACLR analyze gait mechanics less than one year post-ACLR and report fairly reliable results. Quadriceps EMG studies between healthy and ACLR patients across different walking conditions have not been investigated thoroughly in the literature.

Christensen *et al.*[46] investigated inter-limb mechanical asymmetry in subjects six months after receiving TKA. The results concluded significantly greater combined limb support moment, knee extensor moment, and vertical ground reaction force differences during decline walking compared to level walking in patients with TKA.[46] Greater limb support moment, knee extensor moment, and knee joint angle differences were present in patients with TKA compared to healthy-matched peers during decline walking.[46] However, it is not well understood how these different walking conditions may contribute to quadriceps activation, as well as the development of PTOA over time. ACLR knees have been found to have significantly lower peak flexion and extension moments during stair ascent and descent compared to the contralateral knees.[21] This may be due to the contralateral knees compensating for lack stability, muscular strength, proprioception.[21] Identification of quadriceps activation changes in ACLR individuals may allow clinicians to change rehabilitation strategies to create better outcomes for patients in the short term and long term.

Measures of self-report function after ACLR

Understanding this complex issue is also important as even self-reported outcomes measures are sensitive to quadriceps strength deficits. The International Knee Documentation Committee self-reported questionnaire (IKDC) is used as a global assessment of self-reported knee-joint function. Those who scored higher on the IKDC have an increased likelihood of presenting with greater involved limb quadriceps strength and better limb symmetry compared to patients with lower knee function scores after ACLR.[47,48] Furthermore, knee muscle strength has been shown to be a determinant for self-report outcome measures in regards to OA.[49] This has been measured using the

Western Ontario McMaster Universities Osteoarthritis Index (WOMAC) which assesses an individual's perspective of his/her own level of mobility. Berger *et al.*[50] found that OA participants that reported greater functional deficits tended to have lower maximum voluntary quadriceps torque and power. Understanding clinical severity of the disease through self-reported outcome measures and how they relate to objective measures of strength may be critically important in treatment and rehabilitation decisions in patients with OA and potentially ACLR patients.

Conclusions

The quadriceps muscles are important shock-absorbers during gait. Quadriceps dysfunction may be an integral part in the development of PTOA.[11] Dysfunction can involve decreased strength, neuromuscular capability, and coordination and can be a result of muscle atrophy or autogenic inhibition following ACLR. Quadriceps weakness likely contributes to altered gait biomechanics after ACLR.[15] This may cause altered joint loading which is a key mechanism that may contribute to the early development of PTOA.[10,12] Abnormal loading places excess stress on the articular cartilage of the knee, potentially causing earlier degeneration. Dysfunction of these muscles may therefore have significant, life-long effects on ACLR patients.

In healthy individuals, during both upslope and downslope walking the magnitude of rectus femoris and vastus medialis electrical activity significantly increases.[31,42] In terms of timing of muscle activity, the burst durations of the rectus femoris and vastus medialis significantly increase during upslope and downslope walking compared to level walking as well.[31] Individuals ambulate on level and sloped grades daily. Therefore, analyzing how the quadriceps function in individuals with ACLR is important in understanding knee PTOA.[12]

Quadriceps EMG activity may be altered after ACLR and be a significant factor that alters gait biomechanics. Hart *et al.*[12] speculate that altered quadriceps/hamstring function may be related to lower knee flexion angle and moments that occur greater than 6 months after ACLR surgery. As stated by Franz and Kram,[42] understanding how leg muscle activation and gait biomechanics change with grade and speed can help direct therapy in those with limitations in walking. Comparing differences between healthy limb and ACLR limb biomechanics within the same individual or between individuals is important as it may help identify causes and effects of OA after ACL injury.[2]

This study aims to investigate another potential contributing factor to this important issue and help health professionals get a better understanding of gait biomechanics, especially in individuals greater than one year post-ACLR. Individuals one year out from ACLR have gait patterns that seem to normalize over level ground. However, level walking conditions may not be enough to exacerbate the aberrancies that are still present. Lingering abnormalities in gait biomechanics may affect joint loading mechanics which may contribute to developing PTOA. It is important to understand how activities of daily living, particularly repetitive activities such as walking on a variety of surfaces are affected by ACLR. Clinician awareness of the risk of developing PTOA and gait abnormalities in ACLR individuals is crucial for treatment and rehabilitation. Rehabilitation may need to be continued for a longer period of time or be modified to address gait abnormalities. Clinicians may be able to guide treatment to not only address the physical limitations, but also to improve the quality of life.

CHAPTER 3

METHODS

Design

A convenience sample of 51 volunteers participated in this case-control investigation. *A priori* power suggested a sample size of n=32 per group would provide a power of 0.80 with a significance level of 0.05 based on the results from Christensen *et al*.[46] Originally, the sample consisted of 48 subjects per group, but equipment error necessitated exclusion of subjects in both cohorts. The final sample consisted of two cohorts including healthy controls (n=27) and individuals with ACLR (n=24). Male and female subjects between 18 and 35 years of age were recruited. Inclusion criteria for ACLR participants included being at least one year removed from primary unilateral ACLR, no complications from surgery (e.g. revision), no other lower extremity injury in the 6 months prior to participation, and no other history of lower extremity surgery. Inclusion criteria for healthy subjects included no history of lower extremity surgery, knee injury, or lower extremity injury in the 6 months prior to participation. The study was approved by the University's Institutional Review Board and all participants provided written informed consent prior to participation.

Procedures

All data was collected during a single testing session. Gait biomechanics were assessed under 3 conditions (level, uphill, and downhill), and isometric quadriceps function was assessed via an isokinetic dynamometer. The order of assessments for the gait conditions was determined by a balanced Latin square. The order of gait biomechanics and quadriceps function testing was also counterbalanced. All data was assessed bilaterally in both cohorts.

Gait Biomechanics Assessment

Gait biomechanics were collected in a laboratory setting using an 8-camera motion capture system (Qualisys Motion Capture System, Qualisys, Göteborg Sweden) integrated with a split-belt instrumented treadmill (Bertec, Columbus, OH) and EMG system (Trigno, Delsys, Natick, MA). Gait was assessed under level, uphill (10° grade), and downhill (-10° grade) conditions[46] and the speed was set to match the subject's preferred overground speed. Preferred walking speed was determined by having subjects walk overground through infrared timing gates and the average speed across five trials was programmed into the treadmill as a constant for all three conditions. Subjects walked shod for two minutes during each condition wearing an external harness to ensure safety. The first minute was a familiarization period while gait biomechanics were collected during the second minute. Ground reaction forces, marker trajectories, and EMG signals were synced via an external trigger.

A total of twelve surface EMG electrodes were placed bilaterally over the vastus medialis, vastus lateralis, medial and lateral hamstrings, as well as medial and lateral gastrocnemii in order to measure amplitudes of muscle activation. Electrodes were placed over the area of greatest muscle bulk for each muscle determined by manual muscle test, and these sites were shaved, lightly abraded, and cleaned with isopropyl alcohol to improve signal quality and adherence to the skin. EMG electrodes and reflective motion capture markers were attached to the skin with hypoallergenic tape. EMG and ground reaction forces were sampled at 2,000 Hz. Force data were low pass filtered at 10 Hz and used to determine the phases of gait. The stance phase was defined as the interval between heel strike (vGRF > 20N) and toe off (vGRF < 20N). The EMG signals were corrected for DC bias, notch (59.5-60.5 Hz) and bandpass (20-350 Hz) filtered using a 4^{th} order Butterworth filter, and rectified with a root-mean squared approach using a 20ms moving average to create a linear envelope. Quadriceps EMG amplitude, both vastus medialis and vastus lateralis, were the variables of interest and included the mean amplitude over 1) the 200ms prior to heel strike (EMGP), 2) the 200ms centered at heel strike (EMGC), and 3) the first 50% of stance (EMG50%). Each variable was averaged over the first 10 stance phases for each limb. Signals were normalized to the average of subjects' peak EMG amplitude over three maximal voluntary isometric contractions (MVIC). Vastus medialis and vastus lateralis EMG amplitudes were then averaged together to create a composite quadriceps EMG amplitude for each EMGP, EMGC, and EMG50% value that was used in all analyses.

Quadriceps Function Assessment

Subjects were seated on an isokinetic dynamometer (HUMAC Norm, CSMi, Stoughton, MA) with the hip and knee of the tested limb flexed to 90°. The sagittal knee joint axis of rotation was aligned with dynamometer axis of rotation. The testing arm of the dynamometer arm was placed one inch proximally to the malleoli. Padded straps over the torso were used to secure the subject to the seat. All trials were completed with the subject's arms crossed over the chest. EMG electrodes were placed bilaterally over the vastus lateralis and vastus medialis as described above and were not removed between quadriceps function and gait assessments. Subjects performed three warm up trials at 25%, 50%, and 75% of maximal voluntary isometric effort and three maximal trials of isometric knee extension on each limb. Subjects were allowed additional warm up trials if necessary. Once the subject felt comfortable with the testing protocol, he/she performed three recorded maximal effort isometric knee extension trials in which he/she extended the knee maximally and as rapidly as possible. The researcher provided verbal encouragement throughout the trials. Quadriceps function was assessed bilaterally in a randomized order. Quadriceps EMG activity and torque were sampled at 2,000 Hz. Torque was low-pass filtered at 50 Hz (4^{th} order Butterworth digital filter). The EMG signal was filtered identically as described for the gait biomechanics procedures, and the peak value (i.e. the largest 20ms root mean square mean) was extracted from each trial. Peak torque (PT) was defined as the largest torque value during the trial and averaged across the three MVIC trials. Rate of torque development was calculated as the slope of the torque versus time curve from 1) 20% to 80% peak torque (RTD), 2) 0-100ms (RTD_100), and 3) 100ms-200ms (RTD_200). These time intervals were selected as the quadriceps functions differently during 0-100ms and 100-200ms. During 0-100ms the quadriceps must rapidly absorb force and create extension torque during heel strike, whereas 100-200ms is beginning to advance into the stance phase.

Statistical Analysis

Independent samples t-tests were used to evaluate differences in age, height, weight, and gait speed between the ACLR and healthy control cohorts. Prior to analysis, data were screened for outliers and assessed for normality via the Shapiro-Wilk test. Quadriceps EMG amplitudes were evaluated by a 2x3 (group x condition) mixed-model repeated-measures ANCOVA controlling for gait speed. Significant interaction effects were evaluated post-hoc by observing 95% confidence intervals. The groups were determined to differ significantly for each condition if the 95% confidence intervals did not overlap. EMG amplitude change scores were calculated by subtracting the averaged quadriceps EMG in the level condition from the averaged quadriceps EMG in the uphill and downhill conditions, respectively. The relationship between quadriceps function and quadriceps EMG amplitude change scores from the gait biomechanics assessment was completed by partial Pearson correlations controlling for gait speed and time since ACLR, separately for each index of quadriceps function (PT, RTD, RTD_100, RTD_200).

CHAPTER 4

RESULTS

Subject demographics are provided in Table 1. There was no significant difference in age (*P* = 0.727), height (*P* = 0.646), or gait speed (*P* = 0.091) between groups. However, the average weight of the ACLR group was significantly greater (*P* = 0.033). The mean time post ACLR was 4.2 ± 3.2 years.

Table 1. Average demographic information of subjects. P values are for height and weight, respectively

	Age (yrs)	SD	*P*	Height (m)	SD	*P*	Weight (kg)	SD	*P*	Gait Speed (m/s)	SD	*P*	Years Post-ACLR	SD
ACLR (n=24)	21.21	3.80	0.727	1.75	0.098	0.646	77.5	16.426	0.033	1.32	0.165	0.091	4.23	3.16
Control (n=27)	21.54	2.73		1.73	0.091		69.06	9.59		1.25	0.156		N/A	N/A

Table 1 – Average demographic information for all subjects including age, height, weight, gait speed (m/s), and years post-ACLR. Means, standard deviations, and P values are included.

The group x condition interaction effects were not statistically significant for EMGP (*P = 0.089*, observed power = 0.488, Figure 1) or EMG50% (*P = 0.078*, observed power = 0.622, Figure 2). However, the interaction effect for EMGC was significant (*P = 0.017*, observed power = 0.734, Figure 3). Post-hoc analysis did not reveal significant pairwise comparisons as confidence intervals for each group overlapped, but visual inspection of the data revealed differences between groups. The mean ACLR value was 25% lower in level walking and 20% lower in downhill walking compared to the control group, but was 23% higher in uphill walking. The observed power for these post-hoc comparisons ranged 0.151 - 0.316, suggesting that we were underpowered to observe significant group differences. Therefore, although statistically significant results were not obtained, differences may exist. Descriptive statistics for quadriceps EMG amplitudes between groups are provided in Table 2.

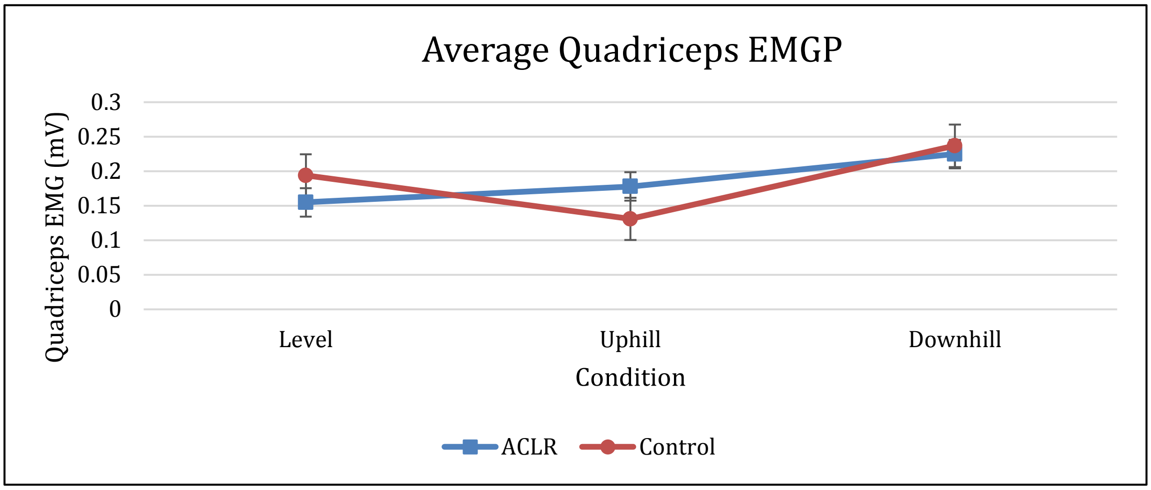

Fig. 1 - The average quadriceps EMG 200ms prior to heel strike for all conditions.

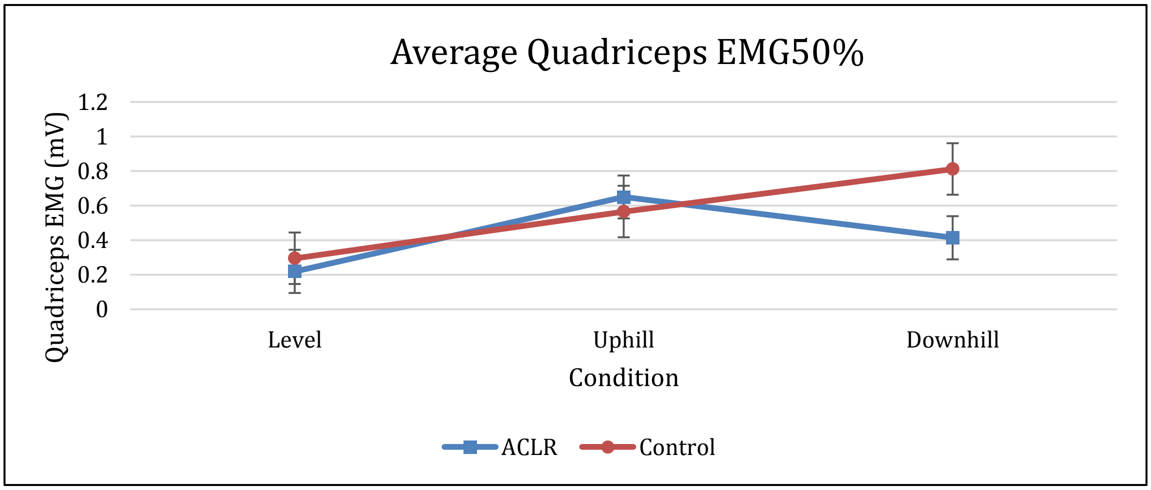

Fig. 2 - The average quadriceps EMG over the first 50% of stance for all conditions.

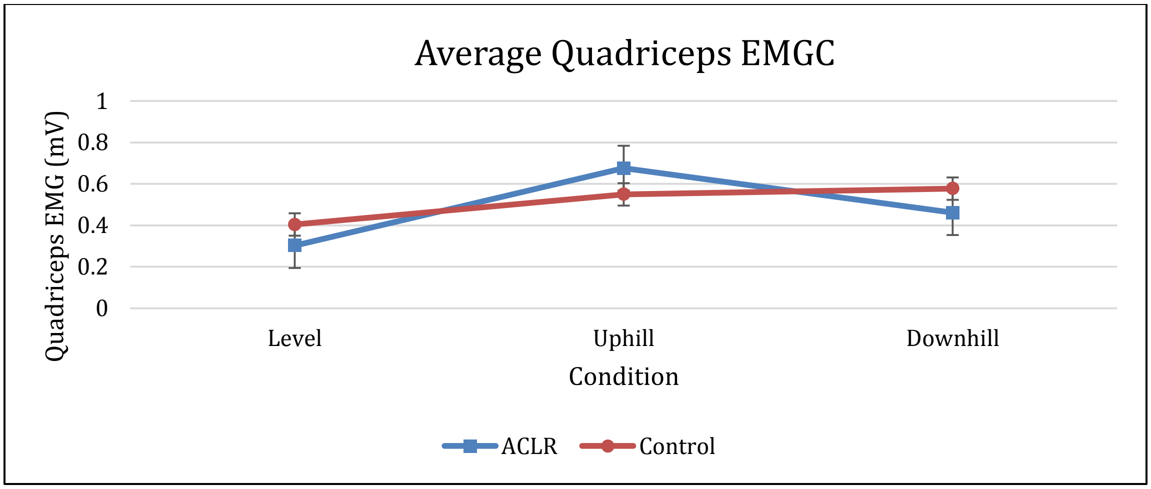

Fig. 3 - The average quadriceps EMG centered at heel strike for all conditions.

Table 2. Estimated marginal means for quadriceps EMG amplitudes between groups

	ACLR (mean ± SE)	CI (range)	Control (mean ± SE)	CI (range)
200 ms prior to heel strike (EMGP)				
Level	0.155 ± 0.038	0.079-0.232	0.194 ± 0.036	0.122-0.266
Uphill	0.178 ± 0.018	0.141-0.215	0.131 ± 0.017	0.096-0.165
Downhill	0.225 ± 0.026	0.174-0.277	0.237 ± 0.024	0.188-0.285
Centered at heel strike (EMGC)				
Level	0.303 ± 0.077	0.148-0.458	0.404 ± 0.072	0.258-0.549
Uphill	0.676 ± 0.066	0.544-0.808	0.550 ± 0.062	0.426-0.674
Downhill	0.461 ± 0.056	0.348-0.573	0.578 ± 0.053	0.473-0.684
Heel strike to 50% stance (EMG50%)				
Level	0.219 ± 0.056	0.106-0.332	0.295 ± 0.052	0.191-0.399
Uphill	0.650 ± 0.058	0.534-0.766	0.565 ± 0.053	0.457-0.672
Downhill	0.414 ± 0.188	0.036-0.793	0.812 ± 0.174	0.462-1.162

Table 2 – Estimated marginal means for quadriceps EMG for each time point and walking condition are listed. The standard error and confidence interval ranges are provided.

The results of all partial correlations are listed in Table 3. A significant correlation was observed between PT of the involved limb and ΔUphill EMGC (*r = -0.508*, *P = 0.016*). The relationship between PT and the ΔUphill EMGP *(r = -0.103, P = 0.648*) and ΔUphill EMG50% (*r = -0.382, P = 0.079*) were not significant. Similarly, the correlations between PT and ΔDownhill EMGP (*r = -0.168, P = 0.456*), ΔDownhill

EMGC ($r = -0.106, P = 0.638$), and ΔDownhill EMG50% ($r = -0.284, P = 0.201$) were not significant.

Table 3. Statistical results of correlations between PT, RTD, RTD_100, and RTD 200 and EMG change scores for each condition and phase of gait. * indicates signifcant results							
		ΔUphill EMGP	ΔUphill EMGC	ΔUphill EMG50%	ΔDownhill EMGP	ΔDownhill EMGC	ΔDownhill EMG50%
PT	*r* value	-0.103	-0.508	-0.382	-0.168	-0.106	-0.284
	P value	0.648	0.016 *	0.079	0.456	0.638	0.201
RTD	*r* value	-0.302	-0.408	-0.278	-0.174	0.102	-0.015
	P value	0.172	0.059	0.210	0.438	0.653	0.948
RTD_100	*r* value	-0.411	-0.668	-0.532	-0.072	0.232	-0.121
	P value	0.057	0.001 *	0.011 *	0.751	0.298	0.591
RTD_200	*r* value	0.079	-0.182	-0.163	-0.300	-0.388	-0.292
	P value	0.726	0.417	0.468	0.175	0.074	0.188

Table 3 – Partial Pearson correlations were used to analyze relationships between torque and EMG change scores and are listed in the above table. Significant findings are marked with an asterisk.

RTD in the involved limb was not significantly correlated with EMG changes for any of the conditions or phases of gait. A significant correlation was observed between the involved limb RTD_100 and ΔUphill EMGC ($r = -0.668, P < 0.001$), as well as ΔUphill EMG50% ($r = -0.532, P = 0.011$). No other significant relationships were observed at RTD_100 or at RTD_200.

CHAPTER 5

DISCUSSION

Our first hypothesis in this study stated that the uphill and downhill conditions would result in greater quadriceps EMG amplitudes compared to level walking, and that the observed increases would be greater in individuals with ACLR compared to compared to healthy controls. A significant interaction between group and condition was only found for the EMGC amplitude, but post-hoc analysis did not reveal any significant pairwise comparisons. Although not statistically significant, for all phases of gait, mean uphill quadriceps EMG amplitude was greater in the ACLR group compared to the control group, but mean downhill and level EMG amplitudes were greater in the control group.

The reason that changes in quadriceps EMG only differed between the groups near heel strike may be due to the fact that this point in the gait cycle requires greater eccentric action absorb impact loading force. Through the end of the swing phase, the quadriceps function to control limb movement and prepare for load acceptance at heel strike.[51] This scenario places greater demands on the quadriceps relative to the swing phase and the first 50% of stance, potentially explaining why group differences were only observed during this interval. Poorer quadriceps function has been associated with greater heel strike transients that are also larger in ACLR subjects compared to healthy controls.[34] Greater heel strike transients have been linked to impulsive loading and cartilage degeneration, particularly at the tibiofemoral joint.[9] Thus, increased EMG activity at heel strike in the ACLR group may indicate a need for greater quadriceps activation to fully prepare for weight acceptance. In order for the ACLR subjects to complete the task of walking uphill, the motor neuron pool must be activated to a greater extent indicating dysfunction of the quadriceps muscles.

ACLR subjects with poorer quadriceps function displayed larger increases in quadriceps EMG amplitude when walking in the uphill condition versus the level condition, partially supporting our hypothesis. However, this effect was not seen in the downhill condition. EMG activity of the vastus lateralis and vastus medialis muscles significantly increases with an increase in grade.[30,31] This again could indicate the necessity of the quadriceps to activate more of the motor neuron pool in order to compensate for the lack of torque production. Previous research has suggested that lesser peak torque of the quadriceps is correlated with smaller internal extension moments.[15] If this is applied to walking uphill, then the quadriceps are not able to function as well to propel the body up an incline. The lack of quadriceps strength to complete the task may require greater motor unit recruitment and action potential production. The resultant increased electrical activity would be reflected via quadriceps EMG measures.

Healthy individuals performing upslope walking display increased in vastus medialis and rectus femoris burst duration and mean EMG activity compared to level walking.[31] This suggests that increased quadriceps activity may be necessary to counteract increased hamstring activity in order to maintain the knee extensor moment, as increased hamstring activity is a result of the necessary increased hip extensor moment when walking uphill.[31] Additionally, with the knowledge that ACLR individuals tend to have greater quadriceps/hamstring co-contraction during the preparatory and heel strike phases of gait,[41] the need to counteract hamstring activity may be even more predominant causing the observed increases in quadriceps EMG activity. Furthermore, unique neuromuscular control strategies also differ between level, uphill, and downhill walking.[27] It may be the case that the method of control for downhill walking does not necessitate as great changes in quadriceps activation as it does for uphill walking.

Lesser rate of torque development over the first 100ms of activation in the ACLR limb was associated with a greater increase in quadriceps EMG amplitude during the uphill condition relative to the level condition. Immediately after heel strike, the quadriceps are required to generate rapid knee extension torque to allow appropriate impact force attenuation.[9,52,53] It is important to note that the first 100ms of stance represents the second half of heel strike in which EMG amplitudes were calculated (centering on heel strike involved the 100ms prior to and the 100ms after heel strike). If the quadriceps are less efficient in producing sufficient and rapid torque, more activation of the muscle is likely required. It has also been suggested that RTD over the first 100ms following onset of contraction may influence gait biomechanics that are linked to an increased risk for PTOA development.[9] Greater impulsive loading rates have been correlated with a lesser RTD over the first 100ms, thereby causing cartilage damage and decreased cartilage thickness.[9] The results of this study would align with this assumption when considering uphill walking conditions in ACLR individuals.

Any significant correlations seen in this study were observed in the uphill condition only. Perhaps this may be due to a greater, more rapid change between propulsion and absorption during uphill gait compared to other walking conditions. When walking uphill, the quadriceps have to produce work to move body weight horizontally, but also to produce positive vertical displacement against the downward pull of gravity. During downhill walking, negative vertical displacement is aided by gravity thereby reducing concentric quadriceps activity at heel strike. Instead, eccentric activity is required immediately post- heel strike. ACLR individuals >24 months out of surgery tended to overload the ACLR-limb during the weight acceptance and propulsion phases of stance.[35] This overloading may cause the quadriceps EMG activity to increase not only due to accepting more load in weight acceptance, but then having to transition to propulsion against the great gravitational and propulsive loads. Furthermore, aberrant gait biomechanics have been shown to be present in ACLR individuals years after surgery.[6,7,12] These abnormalities influence knee angles, moments, and range of motion, thereby affecting how the lower extremity is positioned during all of gait, particularly near heel strike. Quadriceps activity may need to increase during uphill walking to further compensate for suboptimal limb positioning.

There were a number of limitations in this study. The most prominent limitation is the small sample size and limited statistical power. As stated above, our ANCOVA post-hoc analysis revealed an observed power ranging from 0.151 - 0.316, which suggested the study was underpowered. Due to this, we had limited ability to detect significant group differences. Had the study been able to include the full number of intended subjects, there may have been additional significant results not exhibited in the current analyses. Additionally, the subjects in this study were recruited within a university setting, thus many were fairly young and from a localized region. This may make the results less generalizable to other populations, such as those in the general population or those who are older on average. Furthermore, information about the mechanism of injury

(MOI) to the ACL was not documented, but the manner in which subjects were injured may change how the results should be interpreted. Particularly, secondary injuries to other structures in the knee may have had implications in the rehabilitation process thereby influencing quadriceps function over time. Secondary injury may have prolonged decreased quadriceps use causing atrophy, or caused further neuromuscular deficits. Additionally, the MOI would allow researchers to know what environment the injury occurred in. If all subjects in this study had sports related MOIs, then the results may not be generalizable to non-sports related ACL injuries. Finally, another important factor to consider is the variable types and duration of post-surgical rehabilitation the subjects underwent. Subjects likely experienced varying rehabilitation plans, clinicians, resources, and number of therapy sessions that could potentially led to numerous outcomes in the subjects. Therefore, it is unclear if our results apply to all ACLR patients.

A great deal of research has been conducted to determine how rehabilitation should be structured to focus on correcting quadriceps dysfunction. Typically, post-ACLR rehabilitation focuses on knee extension range of motion, quadriceps strengthening, force dissipation, and knee stability. However, the findings of this study suggest that quadriceps dysfunction is still present in ACLR individuals compared to healthy controls, particularly when walking uphill. Perhaps the current methods of rehabilitation are insufficient and do not address this specific circumstance. Clinicians may need to develop new methods to enhance quadriceps function post-ACLR. This may include focusing more on uphill force absorption and the rapid change from concentric to eccentric loading. Anecdotally, ACLR rehabilitation focuses more so on downward, gravity dependent stability exercises, such as step down, drop landing, and hopping tasks.

This is logical as many sports involve landing on a level surface from height. Additional therapeutic modalities such as electrical stimulation or whole body vibration (WBV) may be useful in improving quadriceps torque and function.[54,55] Individuals with ACLR that underwent WBV have been shown to have increases in peak torque, quadriceps strength, and quadriceps EMG activity.[56,57] Enhanced reflexive muscle activity and corticomotor excitability are thought to cause these outcomes, and may be important factors to consider in the rehabilitation process.[56,57] Current rehabilitation methods do not focus much on tasks that involve an incline, presumably because this is less realistic in the sports world where most ACLR injuries occur. Involving more uphill specific exercises throughout that rehabilitation process may help to combat lingering quadriceps dysfunction issues.[30]

Current research has produced ambiguous results on how gait biomechanics are affected years after ACLR, and scarcely any research regarding quadriceps function during graded gait post-ACLR has been conducted. In this study, even multiple years after surgery, uphill walking was shown to provide a unique challenge to ACLR individuals that was not observed in level walking. Increases in quadriceps EMG values were observed, suggesting that the gait biomechanics during incline walking are influenced by quadriceps function. This finding indicates that individuals with ACLR still function with alterations in quadriceps function years after surgery and this may impact knee loading and force attenuation, further implicating the pattern of PTOA development in such individuals. Future research should expand on the results of this study by more fully exploring how the quadriceps function throughout different walking conditions in order to create a better understanding to PTOA development.

REFERENCES

1. Padua DA, Distefano LJ, et al. National athletic trainers' association position statement: Prevention of anterior cruciate ligament injury. *J Athl Train*. 2018;53(1):5-19.

2. Kaur M, Ribeiro DC, Theis J-C, Webster KE, Sole G. Movement patterns of the knee during gait following ACL reconstruction: A systematic review and meta-analysis. *Sports Med*. 2016;46(12):1869-1895.

3. Hewett TE, Myer GD, Ford KR, et al. Biomechanical measures of neuromuscular control and valgus loading of the knee predict anterior cruciate ligament injury risk in female athletes: A prospective study. *Am J Sports Med*. 2005;33(4):492-501.

4. Mather III RC, Koenig L, Kocher MS, et al. Societal and economic impact of anterior cruciate ligament tears. *J Bone Joint Surg Am*. 2013;95:1751-1759.

5. Luc B, Gribble PA, Pietrosimone BG. Osteoarthritis prevalence following anterior cruciate ligament reconstruction: A systematic review and numbers-needed-to-treat analysis. *J Athl Train*. 2014;49(6):806-819.

6. Khandha A, Manal K, Wellsandt E, Capin J, Snyder-Mackler L, Buchanan TS. Gait mechanics in those with/without medial compartment knee osteoarthritis 5 years after anterior cruciate ligament reconstruction. *J Orthop Res*. 2017;35(3):625-633.

7. Wellsandt E, Gardinier ES, Manal K, Axe MJ, Buchanan TS, Snyder-Mackler L. Decreased knee joint loading associated with early knee osteoarthritis after anterior cruciate ligament injury. *Am J Sports Med*. 2016;44(1):143-151.

8. Andriacchi TP, Koo S, Scanlan SF. Gait mechanics influence healthy cartilage morphology and osteoarthritis of the knee. *J Bone Joint Surg Am - Ser A*. 2009;91(Suppl.1):95-101.

9. Blackburn JT, Pietrosimone B, Harkey MS, Luc BA, Pamukoff DN. Quadriceps function and gait kinetics after anterior cruciate ligament reconstruction. *Med Sci Sports Exerc*. 2016;48(9):1664-1670.

10. Hart JM, Pietrosimone B, Hertel J, Ingersoll CD. Quadriceps activation following knee injuries: A systematic review. *J Athl Train*. 2010;45(1):87-97.

11. Blackburn JT, Pietrosimone B, Harkey MS, Luc BA, Pamukoff DN. Inter-limb differences in impulsive loading following anterior cruciate ligament reconstruction in females. *J Biomech*. 2016;49(13):3017-3021.

12. Hart HF, Culvenor AG, Collins NJ, et al. Knee kinematics and joint moments during gait following anterior cruciate ligament reconstruction: A systematic review and meta-analysis. *Br J Sports Med*. 2016;50(10):597-612.

13. Ewers BJ, Dvoracek-Driksna D, Orth MW, Haut RC. The extent of matrix damage and chondrocyte death in mechanically traumatized articular cartilage explants depends on rate of loading. *J Orthop Res*. 2001;19(5):779-784.

14. Ewers B., Jayaraman V., Banglmaier R., Haut RC. Rate of blunt impact loading affects changes in retropatellar cartilage and underlying bone in the rabbit patella. *J Biomech*. 2002;35(6):747-755.

15. Lewek M, Rudolph K, Axe M, Snyder-Mackler L. The effect of insufficient quadriceps strength on gait after anterior cruciate ligament reconstruction. *Clin Biomech*. 2002;17(1):56-63.

16. Bulgheroni P, Bulgheroni MV, Andrini L, Guffanti P, Giughello A. Gait patterns after anterior cruciate ligament reconstruction. *Knee Surgery, Sport Traumatol, Arthrosc*. 1997;5(1):14-21.

17. Ciccotti MG, Kerlan RK, Perry J, Pink M. An electromyographic analysis of the knee during functional activities. II. the anterior cruciate ligament-deficient and -reconstructed profiles. *Am J Sports Med*. 1995;23(4):515-516.

18. Noehren B, Wilson H, Miller C, Lattermann C. Long term gait deviations in anterior cruciate ligament reconstructed females. *Med Sci Sport Exerc*. 2013;45(7):1340-1347.

19. Gao B, Zheng N(N). Alterations in three-dimensional joint kinematics of anterior cruciate ligament-deficient and -reconstructed knees during walking. *Clin Biomech*. 2010;25(3):222-229.

20. Patterson MR, Delahunt E, Sweeney KT, Caulfield B. An ambulatory method of identifying anterior cruciate ligament reconstructed gait patterns. *Sensors (Switzerland)*. 2014;14(1):887-899.

21. Zabala ME, Favre J, Scanlan SF, Donahue J, Andriacchi TP. Three-dimensional knee moments of ACL reconstructed and control subjects during gait, stair ascent, and stair descent. *J Biomech*. 2013;46(3):515-520.

22. Bush-Joseph CA, Hurwitz DE, Patel RR, et al. Dynamic function after anterior cruciate ligament reconstruction with autologous patellar tendon. *Am J Sports Med*. 2001;29(1):36-41.

23. Hall M, Stevermer CA, Gillette JC. Gait analysis post anterior cruciate ligament reconstruction: Knee osteoarthritis perspective. *Gait Posture*. 2012;36(1):56-60.

24. Butler RJ, Minick KI, Ferber R, Underwood F. Gait mechanics after ACL reconstruction: Implications for the early onset of knee osteoarthritis. *Br J Sports Med*. 2009;43(5):366-370.

25. Patterson MR, Delahunt E, Caulfield B. Peak knee adduction moment during gait in anterior cruciate ligament reconstructed females. *Clin Biomech*. 2014;29(2):138-142.

26. Sanford BA, Zucker-Levin AR, Williams JL, Mihalko WM, Jacobs EL. Principal component analysis of knee kinematics and kinetics after anterior cruciate ligament reconstruction. *Gait Posture*. 2012;36(3):609-613.

27. Lay AN, Hass CJ, Nichols TR, Gregor RJ. The effects of sloped surfaces on locomotion: A kinematic and kinetic analysis. *J Biomech*. 2006;39(9):1621-1628.

28. Silverman AK, Wilken JM, Sinitski EH, Neptune RR. Whole-body angular momentum in incline and decline walking. *J Biomech*. 2012;45(6):965-971.

29. Wall-Scheffler CM, Chumanov E, Steudel-Numbers K, Heiderscheit B. EMG activity across gait and incline: The impact of muscular activity on human morphology. *Am J Phys Anthropol*. 2010;143(4):601-611.

30. Lange GW, Hintermeister RA, Schlegel T, Dillman CJ, Steadman JR. Electromyographic and kinematic analysis of graded treadmill walking and the implications for knee rehabilitation. *J Orthop Sports Phys Ther.* 1996;23(5):294-301.

31. Lay AN, Hass CJ, Richard Nichols T, Gregor RJ. The effects of sloped surfaces on locomotion: An electromyographic analysis. *J Biomech*. 2007;40(6):1276-1285.

32. Blagojevic M, Jinks C, Jeffery A, Jordan KP. Risk factors for onset of osteoarthritis of the knee in older adults: A systematic review and meta-analysis. *Osteoarthr Cartil*. 2010;18(1):24-33.

33. Richmond SA, Fukuchi RK, Ezzat A, Schneider K, Schneider G, Emery CA. Are joint injury, sport activity, physical activity, obesity, or occupational activities predictors for osteoarthritis? A systematic review. *J Orthop Sports Phys Ther*. 2013;43(8):515-524.

34. Co FH, Skinner HB, Cannon WD. Effect of reconstruction of the anterior cruciate ligament on proprioception of the knee and the heel strike transient. *J Orthop Res*. 1993;11(5):696-704.

35. Pietrosimone B, Seeley MK, Johnston C, Pfeiffer SJ, Spang JT, Blackburn JT. Walking ground reaction force post-ACL reconstruction: Analysis of time and symptoms. *Med Sci Sports Exerc*. 2019;51(2):246-254.

36. Miyazaki T, Wada M, Kawahara H, Sato M, Baba H, Shimada S. Dynamic load at baseline can predict radiographic disease progression in medial compartment knee osteoarthritis. *Ann Rheum Dis*. 2002;61(7):617-622.

37. Elias ARC, Hammill CD, Mizner RL. Changes in quadriceps and hamstring

cocontraction following landing instruction in patients with anterior cruciate ligament reconstruction. *J Orthop Sports Phys Ther*. 2015;45(4):273-280.

38. Tsai LC, McLean S, Colletti PM, Powers CM. Greater muscle co-contraction results in increased tibiofemoral compressive forces in females who have undergone anterior cruciate ligament reconstruction. *J Orthop Res*. 2012;30(12):2007-2014.

39. Tsai LC, Powers CM. Increased hip and knee flexion during landing decreases tibiofemoral compressive forces in women who have undergone anterior cruciate ligament reconstruction. *Am J Sports Med*. 2013;41(2):423-429.

40. Saxby DJ, Bryant AL, Modenese L, et al. Tibiofemoral contact forces in the anterior cruciate ligament-reconstructed knee. *Med Sci Sports Exerc*. 2016;48(11):2195-2206.

41. Blackburn T, Pietrosimone B, Goodwin JS, Johnston C, Spang JT. Co-activation during gait following anterior cruciate ligament reconstruction. *Clin Biomech*. 2019;67:153-159.

42. Franz JR, Kram R. The effects of grade and speed on leg muscle activations during walking. *Gait Posture*. 2012;35(1):143-147.

43. Franz JR, Lyddon NE, Kram R. Mechanical work performed by the individual legs during uphill and downhill walking. *J Biomech*. 2012;45(2):257-262.

44. Montgomery JR, Grabowski AM. The contributions of ankle, knee and hip joint work to individual leg work change during uphill and downhill walking over a range of speeds. *R Soc Open Sci*. 2018;5(8):180550.

45. Alexander N, Strutzenberger G, Ameshofer LM, Schwameder H. Lower limb joint work and joint work contribution during downhill and uphill walking at different inclinations. *J Biomech*. 2017;61:75-80.

46. Christensen JC, LaStayo PC, Mizner RL, et al. Joint mechanical asymmetries during low- and high-demand mobility tasks: Comparison between total knee arthroplasty and healthy-matched peers. *Gait Posture*. 2018;60:104-110.

47. Zwolski C, Schmitt LC, Quatman-Yates C, Thomas S, Hewett TE, Paterno MV. The influence of quadriceps strength asymmetry on patient-reported function at time of return to sport after anterior cruciate ligament reconstruction. *Am J Sports Med*. 2015;43(9):2242-2249.

48. Lepley AS, Pietrosimone B, Cormier ML. Quadriceps function, knee pain, and self-reported outcomes in patients with anterior cruciate ligament reconstruction. *J Athl Train*. 2018;53(4):337-346.

49. Maly MR, Costigan PA, Olney SJ. Determinants of self-report outcome measures in

people with knee osteoarthritis. *Arch Phys Med Rehabil*. 2006;87(1):96-104.

50. Berger MJ, Mckenzie CA, Chess DG, Goela A, Doherty TJ. Quadriceps neuromuscular function and self-reported functional ability in knee osteoarthritis. *J Appl Physiol*. 2012;113:255-262.

51. Liikavainio T, Isolehto J, Helminen HJ, et al. Loading and gait symmetry during level and stair walking in asymptomatic subjects with knee osteoarthritis: Importance of quadriceps femoris in reducing impact force during heel strike? *Knee*. 2007;14(3):231-238.

52. Levinger P, Webster KE, Feller J. Asymmetric knee loading at heel contact during walking in patients with unilateral knee replacement. *Knee*. 2008;15(6):456-460.

53. Verdini F, Marcucci M, Benedetti MG, Leo T. Identification and characterisation of heel strike transient. *Gait Posture*. 2006;24(1):77-84.

54. Sonnery-Cottet B, Saithna A, Quelard B, et al. Arthrogenic muscle inhibition after ACL reconstruction: A scoping review of the efficacy of interventions. *Br J Sports Med*. 2019;53(5):289-298.

55. Goetschius J, Hertel J, Saliba S, Brockmeier SF, Hart JM. The effects of patellar tendon vibration on quadriceps strength in anterior cruciate ligament reconstructed knees. *Phys Ther Sport*. 2019;40:71-77.

56. Pamukoff DN, Pietrosimone B, Lewek MD, et al. Whole-body and local muscle vibration immediately improve quadriceps function in individuals with anterior cruciate ligament reconstruction. *Arch Phys Med Rehabil*. 2016;97(7):1121-1129.

57. Pamukoff DN, Pietrosimone B, Lewek MD, et al. Immediate effect of vibratory stimuli on quadriceps function in healthy adults. *Muscle and Nerve*. 2016;54(3):469-478.